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Search:

L18  



Search History

DATE: Wednesday, December 21, 2005 [Printable Copy](#) [Create Case](#)

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side by side			result set
DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI,TDBD; PLUR=YES; OP=OR			
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<u>L16</u>	L15 and estradiol	2618	<u>L16</u>
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END OF SEARCH HISTORY

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FILE 'HOME' ENTERED AT 15:25:53 ON 21 DEC 2005

=> file medline, biosis, fsta, jicst, wpids, biotechds		
COST IN U.S. DOLLARS	SINCE FILE	TOTAL
	ENTRY	SESSION
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=> s LDL and liposome
L1 254 LDL AND LIPOSOME

=> s 11 and Apo b
L2 11 L1 AND APO B

=> d 12 ti abs ibib tot

L2 ANSWER 1 OF 11 MEDLINE on STN
TI Evaluation of antioxidant and prooxidant activities of bamboo Phyllostachys nigra var. Henonis leaf extract in vitro.
AB Solvent-extracted bamboo leaf extract (BLE) containing chlorogenic acid, caffeic acid, and luteolin 7-glucoside was evaluated in vitro for free radical scavenging and antioxidant activities using a battery of test methods. BLE exhibited a concentration-dependent scavenging activity of DPPH radical. BLE prolonged the lag phase and suppressed the rate of propagation of **liposome** peroxidation initiated by peroxy radical induced by 2,2'-azobis(2-amidinopropane dihydrochloride (AAPH) at 37 degrees C. BLE also prevented human low-density lipoprotein oxidation, mediated by Cu(2+), which was monitored by the lower formation of conjugated diene and fluorescence and a reduced negative charge of **apo-B** protein. Finally, BLE protected supercoiled DNA strand against scission induced by AAPH-mediated peroxy radical. Prooxidant activity of BLE was seen in a Cu(2+)-induced peroxidation of structured phosphatidylcholine **liposome**, indicating catalytic peroxidation due to a relatively high reducing power of BLE. It was concluded that the BLE has both antioxidant activity and prooxidant activity; the antioxidant activity was attributed to free radical scavenging activity, and the prooxidant activity, albeit minor, resulted from the reducing power of plant phenolics in the presence of transitional metal ions.

ACCESSION NUMBER: 2000458826 MEDLINE

DOCUMENT NUMBER: PubMed ID: 10956087

TITLE: Evaluation of antioxidant and prooxidant activities of bamboo Phyllostachys nigra var. Henonis leaf extract in vitro.

AUTHOR: Hu C; Zhang Y; Kitts D D

CORPORATE SOURCE: Food, Nutrition and Health, Faculty of Agricultural Science, University of British Columbia, Vancouver, BC,

Canada.

SOURCE: Journal of agricultural and food chemistry, (2000 Aug) 48 (8) 3170-6.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200009
ENTRY DATE: Entered STN: 20001005
Last Updated on STN: 20001005
Entered Medline: 20000925

L2 ANSWER 2 OF 11 MEDLINE on STN

TI Oxidative interaction of unpaired hemoglobin chains with lipids and proteins: a key for modified serum lipoproteins in thalassemia.
AB We searched for a biochemical explanation to the modification of lipoproteins like low-density lipoproteins (**LDL**) observed in patients with the severe hemolytic anemia beta-thalassemia. Because a large fraction of the **LDL** surface is composed of phospholipids, we first explored the possible involvement of phospholipids in the oxidative interaction of **LDL** with hemoglobin (Hb), using brain extract phospholipid liposomes as a model. The relative binding affinity and oxidative interaction of three hemoglobin variants (intact Hb A and isolated beta- and alpha-chains) with **LDL** and **liposome** were compared. Studies carried out at low pH/ionic strength and under physiological conditions revealed that association of hemoglobin variants with the phospholipid liposomes is driven by electrostatic forces but their binding is not a prerequisite for oxidative interaction. Unlike phospholipid liposomes, **LDL** underwent only a negligible association with the Hb variants under all pH/ionic strength conditions. Nevertheless, **LDL** induced oxidation of Hb variants, mostly alpha-chains. The dissimilar behavior of the liposomes and **LDL** indicated that **LDL** protein **apo B** rather than phospholipids is the actual **LDL** surface component which interacts with the hemoglobin variants. This agrees with the finding that **apo B** protein underwent oxidative crosslinking by the hemoglobin variants among which alpha-chains were most active. We concluded from these results that the ability of hemoglobin to undergo autooxidation is the key to its oxidative reactivity toward **LDL**. The results of the present study indicate that the modified **LDL** particles observed in beta-thalassemia may reflect lipoprotein oxidation by alpha-chains in circulation.

ACCESSION NUMBER: 97428182 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9281309
TITLE: Oxidative interaction of unpaired hemoglobin chains with lipids and proteins: a key for modified serum lipoproteins in thalassemia.
AUTHOR: Altamentova S M; Marva E; Shaklai N
CORPORATE SOURCE: Sackler Faculty of Medicine, Tel-Aviv University, Tel-Aviv, 69887, Israel.
SOURCE: Archives of biochemistry and biophysics, (1997 Sep 1) 345 (1) 39-46.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199710
ENTRY DATE: Entered STN: 19971013
Last Updated on STN: 19971013
Entered Medline: 19971002

L2 ANSWER 3 OF 11 MEDLINE on STN
TI Low-density lipoproteins interact with **liposome**-binding sites on the cell surface.
AB Under physiological conditions significant amounts of low-density lipoprotein **LDL** particles are taken up by cells independently of specific high-affinity **LDL** receptors (**apo-B** receptors). Previously it was established that some cells contain surface sites capable of binding liposomes. We proposed that **liposome**-binding sites could contribute to **LDL** interaction with the cell surface via phospholipid molecules of **LDL** particles. To check this hypothesis we studied the competitive interaction of human **LDL** and DPPC liposomes with mouse embryo fibroblasts depleted of **apo-B** receptors by preliminary incubation with **LDL**. We have found that after removal of the **liposome**-binding sites from cell lamellae these areas of the cell surface lose their ability to bind **LDL**.

ACCESSION NUMBER: 91348212 MEDLINE
DOCUMENT NUMBER: PubMed ID: 1879530
TITLE: Low-density lipoproteins interact with **liposome**-binding sites on the cell surface.
AUTHOR: Galkina S I; Ivanov V V; Preobrazhensky S N; Margolis L B; Bergelson L D
CORPORATE SOURCE: Belozersky Laboratory of Molecular Biology and Bioorganic Chemistry, Moscow State University, USSR.
SOURCE: FEBS letters, (1991 Aug 5) 287 (1-2) 19-22.
Journal code: 0155157. ISSN: 0014-5793.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199109
ENTRY DATE: Entered STN: 19911020
Last Updated on STN: 19911020
Entered Medline: 19910927

L2 ANSWER 4 OF 11 MEDLINE on STN
TI [Apolipoprotein B of plasma lipoproteins incorporated in liposomes: immunological properties and organ distribution when administered to rabbits].
Apolipoprotein B plazmennykh lipoproteidov, vstroennyi v liposomu: immunologicheskie svoistva i raspredelenie mezhdu organami pro vvedenii kroliku.
AB Apolipoprotein B (**apo B**) isolated from low density lipoproteins (**LDL**) was built in phospholipid-cholesterol liposomes, with the lipid/protein ratio being equal to 33:1. Such liposomes preserved their integrity, whereas the constituent **apo B** retained its antigenic properties. After intravenous injection to rabbits the pattern of **apo B** **liposome** distribution among organs was similar to that of **LDL**.
Apo B liposomes may be used for goal-oriented transport of some substances to organs and tissues whose cells have specific receptors for **apo B**-containing lipoproteins.

ACCESSION NUMBER: 84025000 MEDLINE
DOCUMENT NUMBER: PubMed ID: 6615606
TITLE: [Apolipoprotein B of plasma lipoproteins incorporated in liposomes: immunological properties and organ distribution when administered to rabbits].
Apolipoprotein B plazmennykh lipoproteidov, vstroennyi v liposomu: immunologicheskie svoistva i raspredelenie mezhdu organami pro vvedenii kroliku.
AUTHOR: Klimov A N; Korovkin B F; Kuznetsov A S; Popov I N
SOURCE: Biulleten' eksperimental'noi biologii i meditsiny, (1983 Oct) 96 (10) 47-50.

PUB. COUNTRY: Journal code: 0370627. ISSN: 0365-9615.
USSR
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: Russian
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198312
ENTRY DATE: Entered STN: 19900319
Last Updated on STN: 19900319
Entered Medline: 19831217

L2 ANSWER 5 OF 11 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
TI Anti-leishmanial drug delivery: Acetylated **LDL** as a site-specific delivery ligand.

AB The potential utility of acetylated **LDL** incorporated reverse-phase evaporation vesicles as J774.EL macrophage specific delivery system was studied using Pentostam as anti-leishmanial drug and Leishmania mexicana mexicana as model macrophage disease. The investigations have shown that Apoprotein-B moiety of acetylated-**LDL** is incorporated into reverse-phase evaporation vesicles (acetylated-liposomes) allowing exploitation of the targeting properties of apoprotein-B ligand.

Incorporation of apo-B into liposome

carriers significantly enhances their uptake by Leishmania infected macrophages via the **LDL** and acetylated **LDL** receptors.

The leishmanicidal action of Pentostam entrapped in acetylated liposomes was greater than native **LDL** containing liposomes and significantly higher than untargeted liposomes. Indeed targeted liposomes with acetylated **LDL** ligand have highly beneficial effect on the anti-leishmanial action of entrapped drugs and could contribute to a reduction in toxicity and increase in therapeutic index of currently prescribed anti-leishmanial drugs.

ACCESSION NUMBER: 2004:385437 BIOSIS

DOCUMENT NUMBER: PREV200400385943

TITLE: Anti-leishmanial drug delivery: Acetylated **LDL** as a site-specific delivery ligand.

AUTHOR(S): Shah, Akram [Reprint Author]; Hart, David

CORPORATE SOURCE: Dept ZoolParasitol Sect, Univ Peshawar, Peshawar, 25120, Pakistan
akramkokab@yahoo.com

SOURCE: Pakistan Journal of Zoology, (2004) Vol. 36, No. 1, pp. 45-52. print.

CODEN: PJZOAN. ISSN: 0030-9923.

DOCUMENT TYPE: Article

LANGUAGE: English

ENTRY DATE: Entered STN: 29 Sep 2004

Last Updated on STN: 29 Sep 2004

L2 ANSWER 6 OF 11 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
TI Methylglyoxal and glyoxal enhance **LDL** oxidation through modification of **apo B** arginyl residues.

ACCESSION NUMBER: 2001:103429 BIOSIS

DOCUMENT NUMBER: PREV200100103429

TITLE: Methylglyoxal and glyoxal enhance **LDL** oxidation through modification of **apo B** arginyl residues.

AUTHOR(S): Mowri, Hiro-Omi [Reprint author]; Keaney, John F.

CORPORATE SOURCE: Boston Univ, Boston, MA, USA

SOURCE: Circulation, (October 31, 2000) Vol. 102, No. 18 Supplement, pp. II.82. print.

Meeting Info.: Abstracts from American Heart Association Scientific Sessions 2000. New Orleans, Louisiana, USA. November 12-15, 2000. American Heart Association.

CODEN: CIRCAZ. ISSN: 0009-7322.

DOCUMENT TYPE: Conference; (Meeting)

Conference; Abstract; (Meeting Abstract)

LANGUAGE: English

ENTRY DATE: Entered STN: 28 Feb 2001

Last Updated on STN: 15 Feb 2002

L2 ANSWER 7 OF 11 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
TI Oxidative interaction of unpaired hemoglobin chains with lipids and
proteins: A key for modified serum lipoproteins in Thalassemia.
AB We searched for a biochemical explanation to the modification of
lipoproteins like low-density lipoproteins (**LDL**) observed in
patients with the severe hemolytic anemia beta-thalassemia. Because a
large fraction of the **LDL** surface is composed of phospholipids,
we first explored the possible involvement of phospholipids in the
oxidative interaction of **LDL** with hemoglobin (Hb), using brain
extract phospholipid liposomes as a model. The relative binding affinity
and oxidative interaction of three hemoglobin variants (intact Hb A and
isolated beta- and alpha-chains) with **LDL** and **liposome**
were compared. Studies carried out at low pH/ionic strength and under
physiological conditions revealed that association of hemoglobin variants
with the phospholipid liposomes is driven by electrostatic forces but
their binding is not a prerequisite for oxidative interaction. Unlike
phospholipid liposomes, **LDL** underwent only a negligible
association with the Hb variants under all pH/ionic strength conditions.
Nevertheless, **LDL** induced oxidation of Hb variants, mostly
alpha-chains. The dissimilar behavior of the liposomes and **LDL**
indicated that **LDL** protein **apo B** rather than
phospholipids is the actual **LDL** surface component which
interacts with the hemoglobin variants. This agrees with the finding that
apo B protein underwent oxidative crosslinking by the
hemoglobin variants among which alpha-chains were most active. We
concluded from these results that the ability of hemoglobin to undergo
autooxidation is the key to its oxidative reactivity toward **LDL**.
The results of the present study indicate that the modified **LDL**
particles observed in beta-thalassemia may reflect lipoprotein oxidation
by alpha-chains in circulation.

ACCESSION NUMBER: 1997:456085 BIOSIS

DOCUMENT NUMBER: PREV199799755288

TITLE: Oxidative interaction of unpaired hemoglobin chains with
lipids and proteins: A key for modified serum lipoproteins
in Thalassemia.

AUTHOR(S): Altamentova, Svetlana M.; Marva, Esther; Shaklai, Nurith
[Reprint author]

CORPORATE SOURCE: Sackler Inst. Mol. Med., Sackler Fac. Med., Tel-Aviv Univ.,
Tel-Aviv 69887, Israel

SOURCE: Archives of Biochemistry and Biophysics, (1997) Vol. 345,
No. 1, pp. 39-46.
CODEN: ABBIA4. ISSN: 0003-9861.

DOCUMENT TYPE: Article

LANGUAGE: English

ENTRY DATE: Entered STN: 27 Oct 1997

Last Updated on STN: 27 Oct 1997

L2 ANSWER 8 OF 11 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
TI LOW-DENSITY LIPOPROTEINS INTERACT WITH **LIPOSOME**-BINDING SITES ON
THE CELL SURFACE.

AB Under physiological conditions significant amount of low-density
lipoprotein **LDL** particles are taken up by cells independently of
specific high-affinity **LDL** receptors (**apo-B**
receptors). Previously it was established that some cells contain surface
sites capable of binding liposomes. We proposed that **liposome**
-binding sites could contribute to **LDL** interaction with the cell
surface via phospholipid molecules of **LDL** particles. To check
this hypothesis we studied the competitive interaction of human

LDL and DPPC liposomes with mouse embryo fibroblasts depleted of **apo-B** receptors by preliminary incubation with **LDL**. We have found that after removal of the **liposome** -binding sites from cell lamellae these areas of the cell surface lose their ability to bind **LDL**.

ACCESSION NUMBER: 1991:452391 BIOSIS
DOCUMENT NUMBER: PREV199192097171; BA92:97171
TITLE: LOW-DENSITY LIPOPROTEINS INTERACT WITH **LIPOSOME** -BINDING SITES ON THE CELL SURFACE.
AUTHOR(S): GALKINA S I [Reprint author]; IVANOV V V; PREOBRAZHENSKY S N; MARGOLIS L B; BERGELSON L D
CORPORATE SOURCE: BELOZERSKY LAB MOLECULAR BIOL BIOORGANIC CHEM, MOSCOW STATE UNIV, MOSCOW 119899, MOSCOW, USSR
SOURCE: Febs Letters, (1991) Vol. 287, No. 1-2, pp. 19-22.
CODEN: FEBLAL. ISSN: 0014-5793.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH
ENTRY DATE: Entered STN: 11 Oct 1991
Last Updated on STN: 11 Oct 1991

L2 ANSWER 9 OF 11 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
TI APO LIPO PROTEIN B OF PLASMA LIPO PROTEINS BUILT IN LIPOSOMES IMMUNOLOGIC PROPERTIES AND DISTRIBUTION AMONG ORGANS AFTER ADMINISTRATION TO RABBITS.
AB Apolipoprotein B (**apo B**) isolated from low-density lipoproteins (**LDL**) was built in phospholipid-cholesterol liposomes, with a lipid/protein ratio of 33:1. Such liposomes preserved their integrity; the constituent **apo B** retained its antigenic properties. After i.v. injection to rabbits, the pattern of **apo B** **liposome** distribution among organs was similar to that of **LDL**. **Apo B** liposomes may be used for goal-oriented transport of some substances to organs and tissues whose cells have specific receptors for **apo B** -containing lipoproteins.

ACCESSION NUMBER: 1984:291988 BIOSIS
DOCUMENT NUMBER: PREV198478028468; BA78:28468
TITLE: APO LIPO PROTEIN B OF PLASMA LIPO PROTEINS BUILT IN LIPOSOMES IMMUNOLOGIC PROPERTIES AND DISTRIBUTION AMONG ORGANS AFTER ADMINISTRATION TO RABBITS.
AUTHOR(S): KLIMOV A N [Reprint author]; KOROVKIN B F; KUZNETSOV A S; POPOV I N
CORPORATE SOURCE: DEP BIOCHEM, INST EXP MED, ACAD MED SCI USSR, LENINGRAD, USSR
SOURCE: Byulleten' Eksperimental'noi Biologii i Meditsiny, (1983) Vol. 96, No. 10, pp. 47-50.
CODEN: BEBMAE. ISSN: 0365-9615.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: RUSSIAN

L2 ANSWER 10 OF 11 JICST-EPlus COPYRIGHT 2005 JST on STN
TI Research and development of new medical imaging. 1.9. Research on the localization change of HDL (Apo-A1) and **LDL** (**Apo-B**) in vessel wall and arteriosclerosis lesion.
AB 1) In order to observe of peroxylipid, formation process of the peculiar autofluorescent material structural change of diabetes mellitus and vascular change with fluorescent concentration-change was observed, and the more detailed change of diabetic vascular change was recognized. 2) From composition experiment by **liposome** model membrane and culture smooth muscle cell, there were cell membrane failure and structural change which showed photochemical reaction, and sensitivity increase attributed to the hematoporphyrin which occurred here was clarified.

ACCESSION NUMBER: 1040727302 JICST-EPlus
TITLE: Research and development of new medical imaging. 1.9.
Research on the localization change of HDL (Apo-A1) and
LDL (Apo-B) in vessel wall and
arteriosclerosis lesion.
AUTHOR: MACHIDA MIKI
CORPORATE SOURCE: Nippon Med. Sch.
SOURCE: Nihon Ika Daigaku Haiteku Risachi Senta Kenkyu Hokoku
Heisei 10nen 4gatsu- Heisei 15nen 3gatsu, (2003) pp. 128.
Journal Code: N20041813
PUB. COUNTRY: Japan
DOCUMENT TYPE: Journal; Article
LANGUAGE: Japanese
STATUS: New

L2 ANSWER 11 OF 11 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN
TI Treatment of angina or angina equivalent, e.g. dyspnea, arrhythmia by
administering large liposomes comprised of phospholipids substantially
free of sterols.
AN 2001-080232 [09] WPIDS
AB WO 200069412 A UPAB: 20050211
NOVELTY - A method for treating angina or anginal equivalent comprises
administering a multiplicity of large liposomes comprised of phospholipids
substantially free of sterols is new.

DETAILED DESCRIPTION - A method for treating angina comprises
administering a multiplicity of large liposomes comprised of phospholipids
substantially free of sterols is new.

INDEPENDENT CLAIMS are also included for

(1) a pharmaceutical kit for treating angina or anginal equivalent
comprising a first container having liposomes; and a second container
having anti-anginal drugs other than said liposomes;

(2) a method of perioperative and/ or pre-operative conditioning of a
subject comprising liposomes; and

(3) a method for treating claudication.

ACTIVITY - Cardiant; antiarrhythmic; antianginal; hypotensive;
analgesic; antianemic.

MECHANISM OF ACTION - Angiotensin-converting enzyme(ACE) inhibitor.

USE - To monitor a level of plasma atherogenic lipoprotein, a cardiac
function preferably EKG abnormality, an S-T segment change, arrhythmia, an
assessment of segmental wall motion, blood viscosity, exercise tolerance,
ambulatory EKG monitoring and to treat angina, preferably stable angina,
unstable angina, variant angina, angina pectoris, an anginal equivalent
selected from an ischemic wall motion abnormality, dyspnea, impaired
exercise tolerance, an arrhythmia, a reduced cardiac function, shortness
of breath, fatigue, abdominal distress and referred pain, hypertension,
hyperthyroidism, pulmonary disease, heart failure, hypermetabolic state,
anemia and claudication. (all claimed)

ADVANTAGE - The combination with drugs enhances the intracellular
movement of cholesterol to the cell membrane.

Dwg. 0/28

ACCESSION NUMBER: 2001-080232 [09] WPIDS
DOC. NO. CPI: C2001-022961
TITLE: Treatment of angina or angina equivalent, e.g. dyspnea,
arrhythmia by administering large liposomes comprised of
phospholipids substantially free of sterols.

DERWENT CLASS: B05

INVENTOR(S): GOLDBERG, D; WILLIAMS, K J; GOLDBERG, D I
PATENT ASSIGNEE(S): (TALA-N) TALARIA THERAPEUTICS INC; (ESPE-N) ESPERION LUV
DEV INC; (ESPE-N) ESPERION MERGERCO INC

COUNTRY COUNT: 91

PATENT INFORMATION:

PATENT NO

KIND DATE

WEEK

LA

PG

WO 2000069412 A1 20001123 (200109)* EN 142
 RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL
 OA PT SD SE SL SZ TZ UG ZW
 W: AE AL AM AT AU AZ BA BB BG BR BY CA CH CN CR CU CZ DE DK DM EE ES
 FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS
 LT LU LV MA MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL
 TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW
 AU 2000050053 A 20001205 (200113)
 EP 1183011 A1 20020306 (200224) EN
 R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT
 RO SE SI
 JP 2003508349 W 20030304 (200319) 184
 AU 773385 B2 20040527 (200465)
 AU 2004203419 A1 20040819 (200474) #
 AU 2004203419 A2 20040819 (200510)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2000069412	A1	WO 2000-US12962	20000512
AU 2000050053	A	AU 2000-50053	20000512
EP 1183011	A1	EP 2000-932314	20000512
		WO 2000-US12962	20000512
JP 2003508349	W	JP 2000-617871	20000512
		WO 2000-US12962	20000512
AU 773385	B2	AU 2000-50053	20000512
AU 2004203419	A1	AU 2004-203419	20040727
AU 2004203419	A2	AU 2004-203419	20040727

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2000050053	A Based on	WO 2000069412
EP 1183011	A1 Based on	WO 2000069412
JP 2003508349	W Based on	WO 2000069412
AU 773385	B2 Previous Publ. Based on	AU 2000050053 WO 2000069412
AU 2004203419	A1 Div ex	AU 773385
AU 2004203419	A2 Div ex	AU 773385

PRIORITY APPLN. INFO: US 1999-134140P 19990514; AU
 2004-203419 20040727

=> e halbert, g,au
 E1 1 HALBERSTSMA/BI
 E2 34 HALBERT/BI
 E3 0 --> HALBERT, G,AU/BI
 E4 3 HALBERTI/BI
 E5 4 HALBERTSMA/BI
 E6 1 HALBERTSTADT/BI
 E7 1 HALBERTSTAEDTER/BI
 E8 3 HALBERZEUGNISSE/BI
 E9 5 HALBES/BI
 E10 1 HALBESBERG/BI
 E11 1 HALBESTERN/BI
 E12 3 HALBETASOL/BI

=> e halbert, g/au
 E1 3 HALBERT V A/AU

E2 2 HALBERT W M/AU
E3 0 --> HALBERT, G/AU
E4 2 HALBERTAL E/AU
E5 20 HALBERTHAL E/AU
E6 9 HALBERTHAL M/AU
E7 1 HALBERTHAL M S/AU
E8 7 HALBERTHAL MICHAEL/AU
E9 1 HALBERTHAL MICHAEL S/AU
E10 1 HALBERTHAL MIKI/AU
E11 1 HALBERTHAL R J/AU
E12 2 HALBERTIAS R/AU